WEST Search History

DATE: Friday, September 27, 2002

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,PGPB,JPAB,EPAB,DWPI; PLUR=YES; OP=AND			
L4	L3 and (tea or mushroom or algae or cereal)	16	L4
L3	L1 and cancer$	85	L3
L2	L1 and apoptosis	24	L2
L1	glycerolipid	196	L1

END OF SEARCH HISTORY

SWER 5 OF 8 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 2
ACCESSION NUMBER: 1996:377383 CAPLUS
DOCUMENT NUMBER: 125:83071
TITLE: Relationship between Arachidonate-Phospholipid Remodeling and **Apoptosis**
AUTHOR(S): Surette, Marc E.; Winkler, James D.; Fonteh, Alfred N.; Chilton, Floyd H.
CORPORATE SOURCE: Section on Pulmonary and Critical Care Medicine, Bowman Gray School of Medicine, Winston-Salem, NC, 27157-1054, USA
SOURCE: Biochemistry (1996), 35(28), 9187-9196
CODEN: BICHAW; ISSN: 0006-2960
PUBLISHER: American Chemical Society
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Our previous studies reveal that three structurally distinct inhibitors of the enzyme CoA-independent transacylase, including the antiproliferative alkyllysophospholipid ET-18-O-CH3, induce programmed cell death (**apoptosis**) in the promyelocytic cell line HL-60. The objective of the current study was to better elucidate the mechanism responsible for **apoptosis**. CoA-IT is an enzyme believed to be responsible for the remodeling of long chain polyunsatd. fatty acids like arachidonate between the phospholipids of mammalian cells. The chronic (24-48 h) treatment of HL-60 cells with all three CoA-IT inhibitors resulted in the inhibition of the remodeling of labeled arachidonate from choline- into ethanolamine-contg. phospholipid mol. species. GC-MS anal. of the fatty acids in phospholipids revealed that CoA-IT inhibitor treatment induced a marked loss of arachidonate-contg. phosphatidylethanolamine and an increase in arachidonate-contg. phosphatidylcholine. This redistribution was specific to arachidonate since the mass distribution of linoleic acid in **glycerolipids** was not affected. In spite of the dramatic redistribution of arachidonate, the total cellular arachidonate content was not altered nor was the relative distribution of total phospholipid classes. The increase of arachidonate in phosphatidylcholine was specifically due to an increase in 1-acyl-2-arachidonoyl-sn-glycero-3-phosphocholine species, whereas the loss of arachidonate in PE was from both 1-acyl- and 1-alk-1-enyl-2-arachidonoyl-sn-glycero-3-phosphoethanolamine species. The incubation of cells with exogenous arachidonic acid or ethanolamine did not reverse the inhibition of proliferation induced by CoA-IT inhibitor treatment. Incubation with CoA-IT inhibitors also induced the characteristic cytoplasmic and nuclear changes assocd. with **apoptosis** as assessed by transmission electron microscopy and DNA fragmentation as detd. by flow cytometry. Taken together, these data show that **apoptosis** in HL-60 cells, induced by blocking arachidonate-phospholipid remodeling, is correlated with a redistribution of arachidonate in membrane phospholipids and suggest that such alterations represent a signal which controls the capacity of cells to proliferate.

L7 ANSWER 6 OF 8 MEDLINE
ACCESSION NUMBER: 97119530 MEDLINE
DOCUMENT NUMBER: 97119530 PubMed ID: 8960353
TITLE: "Cross talk" between the bioactive **glycerolipids** and sphingolipids in signal transduction.
AUTHOR: Brindley D N; Abousalham A; Kikuchi Y; Wang C N; Waggoner D W
CORPORATE SOURCE: Signal Transduction Laboratories, Faculty of Medicine, University of Alberta, Edmonton, Canada.
SOURCE: BIOCHEMISTRY AND CELL BIOLOGY, (1996) 74 (4) 469-76. Ref: 61
Journal code: 8606068. ISSN: 0829-8211.
PUB. COUNTRY: Canada
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
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AB Hydrolysis of phosphatidylcholine via receptor-mediated stimulation of phospholipase D produces phosphatidate that can be converted to lysophosphatidate and diacylglycerol. Diacylglycerol is an activator of protein kinase C, whereas phosphatidate and lysophosphatidate stimulate tyrosine kinases and activate the Ras-Raf-mitogen-activated protein kinase pathway. These three lipids can stimulate cell division. Conversely, activation of sphingomyelinase by agonists (e.g., tumor necrosis factor-alpha) causes ceramide production that inhibits cell division and produces **apoptosis**. If ceramides are metabolized to sphingosine and sphingosine 1-phosphate, then these lipids can stimulate phospholipase D and are also mitogenic. By contrast, ceramides inhibit the activation of phospholipase D by decreasing its interaction with the G-proteins, ARF and Rho, which are necessary for its activation. In whole cells, ceramides also stimulate the degradation of phosphatidate, lysophosphatidate, ceramide 1-phosphate, and sphingosine 1-phosphate through a multifunctional phosphohydrolase (the Mg(2+)-independent phosphatidate phosphohydrolase), whereas sphingosine inhibits phosphatidate phosphohydrolase. Tumor necrosis factor-alpha causes insulin resistance, which may be partly explained by ceramide production. Cell-permeable ceramides decrease insulin-stimulated glucose uptake in 3T3-L1 adipocytes after 2-24 h, whereas they stimulate basal glucose uptake. These effects do not depend on decreased tyrosine phosphorylation of the insulin receptor and insulin receptor substrate-1 or the interaction of insulin receptor substrate-1 with phosphatidylinositol 3-kinase. They appear to rely on the differential effects of ceramides on the translocation of GLUT1-and GLUT4-containing vesicles. It is concluded that there is a significant interaction and "cross-talk" between the sphingolipid and **glycerolipid** pathways that modifies signal transduction to control vesicle movement, cell division, and cell death.